PÆDIATRICS.

UNDER THE CHARGE OF

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A Case of Pemphigus Neonatorum.—HOLT (*New York Medical Journal*, February 5, 1898) reports an interesting case of so-called pemphigus of the newborn, which bears out the findings of Strelitz and others, and shows that certain of these cases are simply varieties of sepsis, and are not due to syphilis, to which all such cases have been most generally attributed.

The infant when admitted to the hospital, at the age of nine days, presented many bullæ over its shoulders and lower part of its body. No history of syphilis was obtained in the parents or in two other children. It was brought from a home of great destitution and squalor, and had evidently been neglected from its birth; but in spite of this it was vigorous and well nourished. The body was very dirty. The bullæ were from a quarter of an inch to an inch in diameter; none were present over the back or chest, none upon the feet or hands, and only two upon the face. Some of the more recent lesions were flaccid, and had slightly turbid contents; others had ruptured, showing a deep red base formed by the cutis vera, and still others showed superficial ulceration, and were discharging pus. The navel was normal, but a moderately severe purulent ophthalmia existed, which showed abundant pus organisms, but no gonococci. The contents of one of the bullæ on the neck and of another on the thigh showed pure cultures of the staphylococcus pyogenes aureus.

The condition grew worse; numerous new lesions appeared, and the baby died of exhaustion on the third day after admission.

The lungs showed large areas of atelectasis in both lower lobes, with emphysema anteriorly and many punctate hemorrhages on the surfaces of both lower lobes. The thymus showed punctate hemorrhages, and the liver, spleen, kidneys, and suprarenals were intensely congested, the latter showing also punctate hemorrhages. Cultures from the lungs showed the staphylococcus with the bacterium lactis aerogenes; the spleen and left kidney, the streptococcus longus; and the liver, the streptococcus longus and the staphylococcus. Cultures of the staphylococcus were injected into a mouse, which was found dead at the end of twenty-four hours, yielding the same organism from the heart's blood and at the site of injection.

Acute Nephritis Complicating Mumps.—CHARLES G. KERLEY (*Archives of Pediatrics*, February, 1898) records a case of mumps of moderate severity in a boy of four years. The child was seen for the first time on the third day of the disease, and rest in bed was enjoined. Five days later it was found that the child had been allowed to be up and about the house. His condition was markedly changed. The skin was pale and waxy in appearance, with œdema of the lower lids and adjacent parts, but none elsewhere. Temperature was 103°; the pulse hard and wiry, beating 140, and the skin was hot and dry. No urine had been passed during the previous twenty-four hours, and for two days before the suppression the urine had been high colored and scanty. After appropriate treatment a small quantity of urine passed was deep-red in color and was loaded with albumin, the microscope showing blood in abundance, with granular and hyaline casts. Recovery was very rapid and complete, normal urine being passed on the sixth day of the attack.

The author directs attention to the value of hot saline flushing of the colon in the management of cases of this type, having found this a most efficient means of inducing the kidneys to resume functional activity.

[This very rare instance of renal disease complicating mumps emphasizes the fact that even in the mildest infectious diseases, as has been so recently proven concerning varicella, the condition of the kidneys should always be watched during the course of the disease and in its convalescence.]

The Pseudo-membranous Anginas of Scarlatina.—HIRSCHFELD (*Jahrbuch f. Kinderheilkunde*, Bd. xliv., S. 237) presents a valuable study of 166 cases of scarlatinal angina observed in the service of Professor Heubner. Three groupings were distinguished.

The first group comprised fifty cases, in which the angina was slight, and appeared under the form of isolated or confluent plaques upon the tonsils, and sometimes invading the uvula and pharynx. The false membrane was distinguishable in these cases from that of diphtheria by its viscid consistence and its feeble adherence to the subjacent tissues. The submaxillary ganglia were frequently swollen, and at times reached the dimensions of a nut, but often tumefaction was absent. This angina exercised almost no influence upon the general state and the morbid phenomena belonging to scarlatina. Appearing ordinarily from the first to the third day, it was over in from ten to fifteen days. The treatment was purely symptomatic and not local. All these children recovered.

In the second group, which comprised the cases designated by the author as the malignant form of scarlatinal angina, the prognosis was extremely grave. Of twenty-seven children presenting this form of the complication, not one recovered. These cases were among children varying in age from nine months to three years, most of whom were exhausted by a previous infectious disease (diphtheria, measles, pertussis, rhachitis, syphilis, etc.). The angina was present at the onset, or appeared from one to four days after the beginning of the disease. The false membranes extended rapidly, the breath became fetid, and sanious fluid came from the nostrils. The swelling of the ganglia was enormous, and reached the clavicles, having the appearance of a diffuse phlegmon of the neck, but yielding no pus upon incision. Other manifestations of septicæmia were suppurative otitis, purulent conjunctivitis often with panophthalmitis, suppurative arthritis, broncho-pneumonia, hemorrhagic nephritis, and catarrhal or pseudo-membranous enteritis. The heart and liver showed parenchymatous degeneration, with hypertrophy of the spleen. Treatment seemed without avail.

In the third group of eighty-nine cases, the pseudo-membranous angina was more chronic in its course. It began as a pseudo-membranous angina or as a catarrhal angina, later becoming membranous, the main disease following its regular course. At the end of the first week the fever recurred, and otitis, synovitis, or erysipelas might be found, but more frequently a new swelling of the submaxillary glands developed. With this the throat, which was usually clean, became affected again within twenty-four or forty-eight hours, and symptoms of the malignant form developed, to which the child sometimes succumbed.

In all these groups the angina is due to the scarlatinal poison, and is aggravated in the last two groups by a secondary streptococcic infection. For local treatment the author advises interstitial injections of a solution of carbolic acid of 3 or 5 per cent. strength, half a Pravaz syringeful of the solution being injected twice daily into each tonsil. This treatment, which seemed of no benefit in the acute malignant cases, was, however, successful in fifty-six out of the sixty cases of the chronic form. In no case were symptoms of carbolic-acid poisoning produced by these injections.

PATHOLOGY AND BACTERIOLOGY.

UNDER THE CHARGE OF

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Syringomeylia.—SAXER (*Centralblatt für Allgemeine Pathologie u. Pathologische Anatomie*, 1898, Bd. ix. p. 6) says that in 1892 E. Cramer summarized in the *Centralblatt für Allgemeine Pathologie* those works which had appeared during the year 1891 on the subject of syringomyelia. The conclusions reached by him in regard to the views held at that time on the subject of syringomyelia were as follows: 1. The etiology of syringomyelia is not yet settled. In certain cases anomalies of development and injury seem to play a certain *rôle*. 2. The symptom-complex of syringomyelia can be produced by various lesions, but in most cases the lesion is a simple chronic growth of neuroglia tissue. 3. The lesion is generally localized in the gray matter behind the central canal of the cord. 4. Morvan's disease is clinically and pathologically only a form of syringomyelia. In Morvan's disease the peripheral nerves may also be diseased. 5. The disease may last two or three years (glioma) to thirty or forty years (simple growth of neuroglia tissue). 6. The cavity arises by the breaking down of pathological tissue (usually simple growth of neuroglia tissue). 7. The connection of the central canal with the cavity is secondary. In like manner Saxer reviews 146 articles which have appeared on this subject since 1892. He discusses first *hydromyelia*, in the classical cases a tube-like dilatation of the spinal canal, lined with characteristic epithelium. Variations of this form, however, occur; the epithelium may be wanting over large areas, the central canal may be double, the cavity may be more or less separated from the central canal in places, or the neuroglia tissue around the central canal may be increased. Leyden has drawn the conclusion from such cases that most syringomyelias are hydromyelias dependent on congenital anomalies. Saxer then takes up *syringomyelia,* under which term is understood an elongated cavity in the cord, which is either entirely independent of the normal central canal or is only connected with it accidentally. The pathogenesis of these cavities is various. In the opinion of most writers they arise most frequently in consequence of the breaking down of a primary growth of neuroglia tissue. To this new formation of neuroglia tissue the terms *gliosis, gliomatosis,* and *glioma* have been applied. K. Miura insists on a sharp distinction being made between true malignant glioma and other forms of neuroglia tissue-growth to which the term gliosis should be applied. The indefinite term "gliomatosis" should be replaced by "diffuse glioma" when it refers to an infiltrating glioma, otherwise by "gliosis." Most authors are of the opinion that in syringomyelia the growth of neuroglia tissue is primary, and the cavity secondary through the breaking down of the newly formed tissue. This view is strongly opposed both by Hoffmann and by Schlesinger in their extensive monographs on this subject. They believe that the starting point of these changes is to be found in congenital anomalies of the central canal combined with hydromyelia. This view is not entirely new, but it is now advanced with the support of the latest discoveries in the histogenesis and histology of the central nervous system, particularly the latest results in regard to the developmentof the ependyma and the neuroglia. In order to make the line of argument clear, Saxer summarizes, for the sake of the uninitiated, the discoveries of the last few years in regard to the neuroglia.

According to Hoffmann, the starting point of the disease is to be found in the majority of cases in congenital anomalies of development, as shown by groups of embryonic cells which remain behind the normal central canal along the line of closure of the neural canal. Multiplication of the central canal has a similar significance. From these embryonic remains there arises a growth of neuroglia tissue in which a cavity is usually produced by the breaking down of this newly formed tissue. Hoffmann classifies the cavities of the cord as follows:

1. Hydromyelia.
2. (*a*) Primary (central) gliosis of the cord, with or without hydromyelia.
    1. Without cavity formation.
    2. With cleft and cavity formation.

(*b*) Central gliosis with or without cleft formation, with indefinite symptoms.

The opinion of Hoffmann and Schlesinger, that syringomyelia is connected with congenital anomalies of the cord, previously had many advocates, and their views may now almost be regarded as a settled dogma. Gerlach has tried to prove that the cavity in syringomyelia is lined by connective tissue derived from the pia; but his conclusions are not generally accepted. Certain cases of tube-like cavities in the cord without gliosis have been reported by Schlesinger, Weigert, and others. Weigert claims that the consideration of syringomyelia as softening of a central gliosis has not the shadow of